## AMENDMENTS TO THE SPECIFICATION

At page 17, please replace the description for Figure 21 with the following paragraph:

Figure 21 shows induction of transactivating version of p63 upon UV irradiation. The time course is similar, but not identical, to p53's induction by UV. This is the first demonstration that p63 (and in particular, the transactivating, p53-like, version) can respond to stress signals such as UV/DNA damage. Primary human foreskin keratinocytes were exposed to UV irradiation ($300J/m^2$), then harvested at times indicated. Total RNA was prepared, and RT-PCR on equal amounts of RNA was performed using primers specific for transactivating (TA) or truncated ($\Delta N$) N-terminal domains of p63 (Yang et al., 1998). TA-p63 is not detectable in untreated (unt) keratinocytes, but expression is induced at 7hrs after UV treatment. $\Delta N$-p63 appears to remain unchanged from untreated cells at all time points taken.

At page 17, please replace the description for Figure 22 with the following paragraph:

Figure 22 shows that p63 protein levels, while high in the basal, proliferative/regenerative layer of squamous epithelia, decreases dramatically upon differentiation/maturation of these keratinocytes. This may implicate p63 in ~~differntiation~~ differentiation processes that are important for both oncogenesis and normal development. Primary human foreskin keratinocytes were treated with 10% fetal bovine serum to induce differentiation, and harvested at times indicated. Cells were lysed in RIPA buffer, boiled in Lammeli buffer, and proteins fractionated by SDS gel electrophoresis. Immunoblotting was performed using an anti-p63 mouse monoclonal antibody, 4A4, which recognizes all p63 isoforms identified to date. p63 protein levels in keratinocytes are seen to decrease progressively upon differentiation, as compared to untreated (unt) samples.

At page 17, please replace the description for Figure 23 with the following paragraph:

Figure 23 shows p63 RNA expression in some human cancer cell lines, mostly cervical carcinoma. RT-PCR was performed on total RNA from several human tumor cell lines* using primers specific for transactivating (TA) or truncated (ΔN) N-terminal domains of p63 (see Figure 1), as indicated (*note: all are cervical carcinoma cell lines except U2OS, which is human osteocarcinoma line). Amongst these cell lines, only the ME 180 control cell line showed a TA-p63 transcript. Instead, a majority showed expression of ΔN-p63, which has been demonstrated to act as a dominant negative protein towards both the tumor suppressor p53, as well as transactivating isotypes of p63.

At page 18, please replace the description for Figure 24 with the following paragraph:

Figure 24 just shows p63 RNA expression in some human breast cancer cell lines. It is useful to note that p63 is expressed in these cancers. ΔN versions also strongly expresses in some of these lines. RT-PCR was performed on total RNA from breast cancer cell lines using primers specific for transactivating (TA) or truncated (ΔN) N-terminal domains of p63 (see Figure 1), as indicated. p63 was detected in a majority of cell lines tested, with some specimens containing both transcript isotypes.

At page 18, please replace the description for Figure 25 with the following paragraph:

Figure 25. Shows the results of an electrophoretic shift assay. Electrophoretic mobility shift assays (EMSA) were performed using three separate, $^{32p}$ radiolabeled oligonucelotides: a minimal p53 binding sequence site (PG), a p53 binding site in the p21 promoter (WAF), and a mutant p53 binding site (MG, *Kern et al., 1992) with lystates of 293 human kidney cells

transfected with p53, ΔNp63γ, and Tap63α, and green fluorescent protein (GSP). p53, ΔNp63γ, and Tap63α lysates all yielded significant mobility shifts of both PG and WAF oligonucleotides (highlighted by arrows), while the GFP protein, included as negative control, failed to display a similar shift. None of the lysates showed a shift of the control, non-p53 binding olignucleotide, MG, thus demonstrating the specificity of p53, ΔNp63γ, and Tap63α interactions with the p53-binding sites.